AMENDMENTS TO THE SPECIFICATION

Amendment 1

Please replace the paragraphs beginning at page 5, lines 6 to 25, with the following amended paragraphs:

~~FIG. 1A.~~ FIG. 2A. Sepharose beads bound with purified glutathione S-transferase (GST, lane 2) and GST fusions of Hec1 containing amino acids 56-642 (GST-hsHec1p, lane 3) or 251-618 (GST-15Pst, lane 4) were mixed with in vitro translated, ^{35}S-methionine-labeled Nek2 (lane 1), then washed extensively. The binding complexes were separated by SDS-PAGE, dried, and visualized by autoradiography.

~~FIG. 1B.~~ FIG. 2B. Specific regions of Hec1 bind to Nek2 by yeast two-hybrid assay. Deletion mutants containing the different coiled-coil domains of Hec1 were fused in-frame to a GAL4 DNA binding domain. Nek2 was expressed as a GAL4 transactivation domain fusion. Yeast transformants with these two hybrid proteins were grown in liquid cultures and used for O-nitrophenyl-β-galactopyranosidase quantitation of β-galactosidase activity. The fold-increase in activity compared to the host yeast strain Y153 is indicated. Assays were done in triplicate for each transformation.

~~FIG. 1C.~~ FIG. 2C. Cell cycle-dependent interaction between Hec1 and Nek2. T24 bladder carcinoma cells were first density arrested at G1 (lanes 2) and then released for re-entry into the cell cycle. At different time points after release from density arrest (indicated above the lanes), cells were collected and lysed. The clarified lysates were immunoprecipitated with mAb9G3 anti-Hec1 monoclonal antibodies (upper two panels) or anti-Nek2 antisera (lower two panels). Hec1 and Nek2 co-immunoprecipitated at G2 and M phases (lanes 5 and 6).

Amendment 2

Please replace the paragraph at page 41, lines 8 to 17, with the following amended paragraphs:

To determine whether the identified compounds would have the same effect in

mammalian cells, we used HeLa cells to test the effects of these compounds in a small-scale experiment. With a dose of 20 μM, two of the original eight compounds showed significant activity in killing dividing cells. These two compounds were named INH1 and INH2 (INH1, MW: 308.41; also referred to herein as IBT 13131; see FIG. 16 ~~FIG. 17~~; INH2, MW 382.49; also referred to herein as IBT 14664; see FIG. 16 ~~FIG. 17~~). Interestingly, these two chemicals contain a core *N*-(4-phenylthiazol-2-yl)benzamide ~~phenyl-thiozol-benzamide~~ structure with additional groups on both sides of the benzene rings. The remaining six compounds that showed no activity did not share the same core structure. INH1 is smaller than INH2, and has a significant higher killing activity (FIG. 12) as measured by colony formation assays.